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FILE COVERS 1907 - 27 Oct 2004 VOL 141 ISS 18
FILE LAST UPDATED: 26 Oct 2004 (20041026/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s tocopherol
         27182 TOCOPHEROL
          8038 TOCOPHEROLS
L1       29429 TOCOPHEROL
                (TOCOPHEROL OR TOCOPHEROLS)

=> s l1 and protect?
        520754 PROTECT?
L2        3755 L1 AND PROTECT?

=> s l2 and purif?
        749054 PURIF?
L3           89 L2 AND PURIF?

=> s l3 and hydrolysis
        402747 HYDROLYSIS
          3086 HYDROLYSES
        403584 HYDROLYSIS
                (HYDROLYSIS OR HYDROLYSES)
L4            3 L3 AND HYDROLYSIS

=> s l3 and hydroly?
        570858 HYDROLY?
L5            4 L3 AND HYDROLY?

=> s l3 and solvoly?
         14030 SOLVOLY?
L6            1 L3 AND SOLVOLY?

=> dup rem l6 l5 l4
PROCESSING COMPLETED FOR L6
PROCESSING COMPLETED FOR L5
PROCESSING COMPLETED FOR L4
L7            5 DUP REM L6 L5 L4 (3 DUPLICATES REMOVED)
```

=> d l7 ibib hitstr abs 1-5

L7 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2004:100941 CAPLUS
DOCUMENT NUMBER: 140:151967
TITLE: Preparation of color-stable low impurity **tocopherol** compositions
INVENTOR(S): Milstein, Norman
PATENT ASSIGNEE(S): Cognis Corporation, USA
SOURCE: PCT Int. Appl., 15 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004010931	A2	20040205	WO 2003-US23277	20030725
WO 2004010931	A3	20040624		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2004138479	A1	20040715	US 2003-626281		20030724
PRIORITY APPLN. INFO.:			US 2002-398900P	P	20020726
			US 2003-626281	A	20030724

AB Processes for preparing color-stable, low impurity **tocopherol** compns. are described, wherein the processes comprise: (a) providing a **protecting** group-substituted **tocopherol** compound, for example an acetate of a natural-source **tocopherol** compound; (b) **purifying** the **protecting** group-substituted **tocopherol** compound, e.g., through crystallization; and (c) **solvolyzing** the **purified** compound to form free **tocopherol.** Also described are the **tocopherol** compns. prepared thereby.

L7 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2004:828317 CAPLUS
TITLE: Potential antioxidant peptides in rice wine
AUTHOR(S): Rhee, Sook Jong; Lee, Chung-Yung J.; Kim, Mi-Ryung; Lee, Cherl-Ho
CORPORATE SOURCE: Graduate School of Biotechnology, Korea University, Seoul, 136-701, S. Korea
SOURCE: Journal of Microbiology and Biotechnology (2004), 14(4), 715-721
CODEN: JOMBES; ISSN: 1017-7825
PUBLISHER: Korean Society for Microbiology and Biotechnology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Many food protein **hydrolyzates** have been shown to have antioxidant activities, and recent research focuses on low mol. peptides

produced during **hydrolysis** of food protein. Korean rice wine contains about 60-70% of protein at dry base and originates from raw materials. It has been suggested that the protein is transformed into low mol. weight peptides, and have antioxidant activity during fermentation The objectives of this study were to evaluate the antioxidant activity of the pre-**purified** and **purified** peptides found in Korean rice wine and to identify the responsible peptides. The wine extract of Samhaeju, a traditional Korean rice wine made by low temperature fermentation, was evaporated at 35°C. The two methods employed in the evaluation of antioxidant activity were the DPPH radical scavenging method and the beta-carotene bleaching test. The pre-**purified** samples showed 808 AAC (Antioxidant Activity Coefficient) and 56.5% AOA (Antioxidant Activity), which were higher than α- **tocopherol** (572 AAC and 78% AOA). The rice wine extract was separated by reversed-phase HPLC. The **protective** effect of the four most antioxidant active fractions were tested for t-Bu hydroperoxide induced oxidation of healthy human erythrocytes and the byproduct was determined by malondialdehyde formation. Fraction Number 5 showed 35% lower MDA concentration as compared to the control.

The peptides were further **purified** using consecutive chromatog. methods and 4 antioxidant peptides were isolated. The amino acid sequences of the peptides were identified as Ile-His-His, Val-Val-His(Asn), Leu-Val-Pro, and Leu(Val)-Lys-Arg-Pro. The AAC value of the synthetic form of the identified peptides was the highest for Ile-His-His.

L7 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2004 ACS on STN

ACCESSION NUMBER: 1997:707100 CAPLUS
DOCUMENT NUMBER: 128:32750
TITLE: Evidence for a paraoxonase-independent inhibition of low-density lipoprotein oxidation by high-density lipoprotein
AUTHOR(S): Graham, Annette; Hassall, David G.; Rafique, Samina; Owen, James S.
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Royal Free Hospital School of Medicine, Rowland Hill Street, London, NW3 2PF, UK
SOURCE: Atherosclerosis (Shannon, Ireland) (1997), 135(2), 193-204
CODEN: ATHSBL; ISSN: 0021-9150
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

AB One mechanism by which plasma high-d. lipoprotein (HDL) may **protect** against atherogenesis is by inhibiting the oxidation of low-d. lipoprotein (LDL). Recent evidence suggests that paraoxonase, an HDL-associated, calcium-dependent enzyme, may be responsible for the antioxidant action of HDL (Mackness et al., Atherosclerosis 1993;104:129; Mackness et al., FEBS Lett 1991;286:152; Watson et al., J Clin Invest 1995;96:2882; Navab et al., Arterio Thromb Vasc Biol 1996;16:831); in particular, paraoxonase activity inhibits the formation of 'minimally oxidized' LDL by **hydrolyzing** biol. active oxidized phospholipids (Watson et al., J Clin Invest 1995;96:2882; Navab et al., Arterio Thromb Vasc Biol 1996;16:831). However, antioxidant effects of HDL have also been demonstrated under calcium-free conditions, arguing that this enzyme may not be the only mechanism by which HDL inhibits LDL oxidation (Tribble et al., J Lipid Res 1995;36:2580). Here we have evaluated the role of paraoxonase in prevention of LDL oxidation by using HDL subfractions, isolated from human serum or EDTA-plasma, which display markedly different

levels of paraoxonase activity; the abilities of modified forms of HDL to prevent LDL oxidation by cultured human (THP-1) macrophages were also assessed. Paraoxonase activity was substantially lower in HDL prepared from plasma compared to serum HDL; moreover, virtually all of the lipoprotein-associated paraoxonase activity was located in the HDL3 fraction, with HDL2 retaining only 1-5% of the total activity. Despite possessing 5-fold differences in paraoxonase activity, HDL3 isolated from plasma or serum was equally effective in inhibiting LDL oxidation by THP-1 macrophages; furthermore, although plasma HDL3 was more **protective** than plasma HDL2, the latter did significantly inhibit LDL oxidation Non-paraoxonase antioxidant constituents of plasma HDL3 were investigated further. ApoHDL3, the totally delipidated form of HDL3, was much less effective than native HDL3; when examined individually, **purified** apolipoprotein A-II gave greater **protection** than apo A-I, although this effect was not evident in apo A-II-enriched HDL3. Partial delipidation of HDL3, which removes both neutral lipids and α-**tocopherol**, did not significantly diminish its ability to inhibit LDL oxidation by THP-1 macrophages; phospholipid vesicles prepared from partially delipidated HDL3 also inhibited LDL oxidation effectively. We conclude that, in this model of cellular LDL oxidation, the phospholipid fraction of HDL exerts inhibitory effects which are independent of HDL paraoxonase activity.

REFERENCE COUNT: 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 1991:674331 CAPLUS
DOCUMENT NUMBER: 115:274331
TITLE: Modulation of the activity of hepatic glucose-6-phosphatase by methylthioadenosine sulfoxide
AUTHOR(S): Speth, Maria; Schulze, Hans Ulrich
CORPORATE SOURCE: Biochem. Inst., Justus-Liebig-Univ., Giessen, 6300, Germany
SOURCE: Biochimica et Biophysica Acta (1991), 1068(2), 217-30
CODEN: BBACAQ; ISSN: 0006-3002
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Methylthioadenosine sulfoxide (MTAS), an oxidized derivative of the cell toxic metabolite methylthioadenosine has been used in elucidating the relevance of an interrelationship between the catalytic behavior and the conformational state of hepatic glucose 6-phosphatase and in characterizing the transmembrane orientation of the integral unit in the microsomal membrane. The following results were obtained: (1) glucose 6-phosphate **hydrolysis** at 37° is progressively inhibited when native microsomes are treated with MTAS at 37°. In contrast, glucose 6-phosphate **hydrolysis** of the same MTAS-treated microsomes assayed at 0 °C is not inhibited. (2) Subsequent modification of the MTAS-treated microsomes with Triton X-114 reveals that glucose 6-phosphatase assayed at 37° as well as at 0° is inhibited. (3) Although excess reagent is separated by centrifugation and the MTAS-treated microsomes diluted with buffer before being modified with Triton the temperature-dependent effect on MTAS on microsomal glucose 6-phosphatase is not reversed at all. (4) In native microsomes MTAS is shown to inhibit glucose 6-phosphatase noncompetitively. The subsequent Triton-modification of the MTAS-treated microsomes, however, generates an uncompetitive type of inhibition. (5) Preincubation of native microsomes with MTAS completely prevents the inhibitory effect of 4,4'-diisothiocyanostilbene 2,2'-disulfonate (DIDS) as well as 4,4'-diazidostilbene 2,2'-disulfonate (DASS) on glucose 6-phosphatase. (6) Low mol. weight thiols and **tocopherol protect** the

microsomal glucose 6-phosphatase against MTAS-induced inhibition. (7) Glucose 6-phosphatase solubilized and partially **purified** from rat liver microsomes is also affected by MTAS in demonstrating the same temperature-dependent behavior as the enzyme of MTAS-treated and Triton-modified microsomes. From these results it is concluded that MTAS modulates the enzyme catalytic properties of hepatic glucose 6-phosphatase by covalent modification of reactive groups of the integral protein accessible from the cytoplasmic surface of the microsomal membrane. The temperature-dependent kinetic behavior of MTAS-modulated glucose 6-phosphatase is interpreted by the existence of distinct catalytically active enzyme conformation forms. Detergent-induced modification of the adjacent hydrophobic microenvironment addnl. generates alterations of the conformational state leading to changes of the kinetic characteristics of the integral enzyme.

L7 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 1954:61084 CAPLUS
DOCUMENT NUMBER: 48:61084
ORIGINAL REFERENCE NO.: 48:10866g-i
TITLE: Factors **protecting** against dietary necrotic liver degeneration
AUTHOR(S): Schwarz, Klaus
CORPORATE SOURCE: U.S. Pub. Health Service, Bethesda, MD
SOURCE: Annals of the New York Academy of Sciences (1954), 57, 878-88
CODEN: ANYAA9; ISSN: 0077-8923
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB A recapitulation of work dating from 1940, demonstrating that cystine (I), vitamin E (α- **tocopherol** acetate, II), and Factor 3 (III) **protect** against dietary necrotic liver degeneration in rats. Two necrogenic diets low in I and deficient in II and III are described. Addition of 0.2-1% I to these diets prevents necrosis. Other S-containing amino acids like methionine, homocystine, and cysteine are only 1/3 as effective as I. II affords 50% **protection** at 50-67 γ daily levels per rat, which is within the normal range of II requirement. Detection, occurrence in caseins and brewers' yeast, and **purification** of III are described. This is a low-mol. weight, water-soluble compound which is stable against acid **hydrolysis** and is not identical with known vitamins or amino acids. Study of the metabolic interrelations in dietary liver necrosis suggests a primary metabolic defect closely related to the citric acid cycle. 30 references.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	26.31	26.52

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-3.50	-3.50

STN INTERNATIONAL LOGOFF AT 18:09:48 ON 27 OCT 2004